● *ABSTRACT*

Regulation of new technologies to protect public health and the environment assumes that the regulators have adequate information with which to make informed decisions. In the case of DDT, however, full information on its effects did not become available until two decades after it came into widespread use. This situation arose for a variety of reasons. For some years, scientists lacked analytical tools to measure quickly small concentrations of residues (a minor problem). The organization and funding of science diverted attention from long range basic studies to immediate problems, and DDT seemed to pose no obvious hazard. Most important, past experience proved to be an inadequate guide to research on DDT's effects — a consequence of the novel chemical and physical properties of the material.

Science as a Guide in Regulating Technology:

The Case of DDT in the United States

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During and shortly after World War II, chemists developed a wide variety of chlorinated hydrocarbon insecticides which revolutionized insect control, made major contributions to public health, and changed agriculture throughout the world. Two decades later another group of scientists, mainly biologists, established a strong case that the most important, best known, and most widely used of these compounds, DDT, had properties which had not been uncovered in earlier research. They found that it was persistent, quite mobile, and capable of being concentrated through food chains, and that it caused a variety of effects in non-target organisms. Many scientists believed that it was responsible for reproductive failures in birds and a number of other environmental disturbances. Some considered it a potential human health hazard.[1] Their findings led to both popular and scientific opposition to the continued use of the

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chemical and, in the early 1970s, many countries either banned or severely restricted its use. It has been argued with considerable vigour that the decision to ban DDT was a mistake — that the specific benefits of continued use outweighed the general costs.[2] This is not at issue here. Nor are we concerned with the economic and political dimension of policy or the changes in values which played a large role in the controversy. Our problem is an antecedent one. Why, during most of the period in which DDT was used, did neither policy makers nor the public have the full information needed to make rational cost-benefit decisions?

There are many reasons. One, comparatively minor, was the lack (up to about 1960) of analytical tools which would enable scientists easily to trace pesticide residues through the environment. Only with the development of the vapour-phase chromatograph could scientists quickly handle many samples and test for several compounds at once. Technical equipment, though, while it accelerated the elucidation of DDT's movement through the ecosystem, was not crucial. More important in concealing the full range of effects was the kind of effect that DDT was alleged to have. The case against the chemical was based on its purported, novel effects in the environment, and both the organization and funding of research and the standard scientific disciplinary boundaries hampered work on this type of problem. Chemical companies, which did much of the research on insecticides, had little incentive to support studies on the long range effects of chemicals in the environment; there was no profit in such work and no obligation to do it. Government agencies, chronically short of funds and driven by the annual need to justify their budget, concentrated on immediate problems of some interest to the public. They had to show results, and basic studies, unless directed to an obvious need, were not the way to do it. The problem of DDT residues also cut across disciplinary lines, which complicated research programmes and left scientists, working in normal channels, with only part of the evidence.

There is still a third factor — the lack of an adequate guide to investigation. It is impossible to test for all possible effects of a new chemical; scientists need some indication of where to look. The most useful, often the only, guide is experience with similar compounds or with compounds used in similar situations. In the case of DDT, though, experience with chemicals previously introduced into the environment proved to be an incomplete and misleading guide. Reasoning by analogy and following familiar research strategies

took scientists in the wrong directions. Although there were early warnings about possible problems arising from the use of these new chemicals, no one anticipated the form the effect would take or deduced where to look for them, and this was crucial to the development of the concern over DDT.

An Old Problem: Insecticides and Human Health

The first investigations of DDT's effects on non-target organisms were on humans, and the nature and extent of the chemical's action on man remained the primary concern of government regulation for two decades. This emphasis reflected experience with the arsenical and botanical insecticides, which had been employed largely in agriculture and, hence, posed little danger to wildlife. Assumptions about the chemical's possible effects, about the best way to establish its action in humans, and about the criteria by which safety should be judged limited and directed studies of DDT's effects in man. The ways in which experience and institutional arrangements shaped the investigation of medical scientists can most clearly be seen by considering the formation of policy on insecticide residues and the evidence cited as proof of DDT's safety to man.

The most prominent American authority on the subject of DDT in humans was Dr Wayland J. Hayes, Jr., a Public Health Service physician who had begun work on the compound in the late 1940s. Hayes was a key witness and advocate for the pro-DDT forces, and appeared at hearings to defend the safety of the chemical. The defenders of DDT frequently, and approvingly, cited his work. Hayes believed that DDT was safe for the general population over a lifetime of exposure; his evidence was derived from studies on the health of workers in DDT manufacturing plants, men who had prolonged contact with the chemical, and of convict volunteers who had ingested high doses of DDT daily for periods of up to a year.[3]

A 1958 study of occupational exposure, for example, had included men who had worked in DDT plants for up to 6½ years; one conducted in 1967 had included men with up to 19 years' experience. Although these men had much more exposure to DDT than the general population and much higher levels of DDT in their fat, none showed symptoms of DDT poisoning or had any pathological conditions attributable to the chemical. Convict volunteers had ingested doses of up to 3.5 milligrams per day for a year. DDT levels in their

fat had ranged, in two studies, up to 234 and 281 parts per million (ppm), in contrast to a level in the population of 2.3-4.0 ppm. They, too, had shown no pathological symptoms, even in checkups two years after the end of the experiment.[4]

The tests, and the extrapolation from them to the general population, involved certain assumptions about human health and chemical poisoning. They took for granted that the only important effects of DDT on the human body would be those shown in acute poisoning — damage to the central nervous system and to the liver; the only tests done on the subjects had been to identify gross neurological changes and clinical damage to body functions. The researchers implicitly accepted the concept of a 'threshold' — a 'no-effect'level — below which the chemical would produce no changes in the body. Using the test groups as an indication of the potential effects of the chemical in the general population called for further assumptions. One was that deleterious effects on the general population could best be discovered by studying humans with a higher than normal exposure to the chemical (rather than studying experimental animal populations); another, that these self-selected groups were adequate samples of the population.[5]

Concern about these standards was not a product of the 1960s or even of the introduction of the chlorinated hydrocarbon insecticides. The basis of American federal residue policy had been established in the period between the early 1920s, when the government first began to consider insecticide residues on fruit a possible health hazard, and 1940, when the last changes in the pre-DDT standards had been made.

The apple growers, the major force behind the fight for high tolerance levels in the United States, had cited as proof of safety the lack of human death and illness produced by eating sprayed apples, and they decried the usefulness of animal studies for evaluating hazards to humans. Private physicians and the Food and Drug Administration, on the other hand, warned of the possibility of chronic damage and stressed the lack of information on the effects of low doses over a long period. During the Depression the growers found a sympathetic audience in Congress. Regulation bowed to the growers' needs: studies made before the introduction of DDT emphasized the criteria favoured by the farmers — clinical symptoms of poisoning and studies of exposed human populations[6] — and relied on an agency, the Public Health Service, which agreed with these assumptions.

The American debate over DDT in the early years followed the

same scenario and had the same result. Almost the only change was the replacement of the International Apple Association with the National Agricultural Chemicals Association as the most prominent defender of the standards. There were early warnings about the possible dangers of chronic doses of the new chemical, and about the lack of data in this area, and recommendations for further research.[7] As in the 1930s the two sides met before Congress, where the arguments over the criteria to be used in setting standards for residues of DDT on food and in assessing the possible dangers of the chemical were the subject of testimony before the House Select Committee to Investigate the Use of Chemicals in Food Products during 1950 and 1951.

By this time scientists knew that most American meals contained DDT, that virtually the entire population was storing the chemical in body fat, and that even small doses affected liver function in rats. What, though, was the significance of the data? There was a clear difference of opinion. Scientists from the Public Health Service and the Department of Agriculture, strongly supported by the manufacturers and users of the chemical, thought that DDT was safe. They claimed that war-time experience and tests had shown that people could be exposed without harm, and continuing research would be adequate to safeguard the public from any long-term effects that might show up. Tests, they said, would be made on populations exposed to higher-than-normal levels of the chemical. However, pharmacologists and physicians from the Food and Drug Administration and various private groups thought that there was a potential hazard from chronic exposure to DDT; they urged long term testing of experimental animals and the extrapolation of these results as a guide to regulatory policy. Representatives of the National Cancer Institute, the American Cancer Society, and various consumer groups concurred. The widespread use of DDT, they said, meant that many people not in good health or unusually susceptible to damage, such as infants, were being exposed. They urged tighter control and restrictions on DDT's use. Oncologists were particularly concerned about the long-term effects, citing the latency period of many carcinogens.[8]

As a result of these hearings Congress made changes in the laws regulating pesticides and pesticide residues on food. The Miller amendment to the 1938 Food, Drug and Cosmetic Law (68 US Stat. 511), passed in 1954, placed the burden of proof of safety of a pesticide on the user. Another, the Delaney Amendment (21 USC

sec. 348 (c) (3) (A)), required a zero tolerance on any chemical known to cause cancer in any experimental animal or in man. Congress did not, however, require long-term testing of the chronic effects of new agricultural chemicals on man or on experimental animal populations, and generally accepted tests on exposed human populations as an indication of the risk to the general population.

Economic pressures cannot explain general acceptance of these standards, but neither the Congress, nor the public, nor any significant group of toxicologists or physicians pressed for long-range, detailed studies of DDT's effects. One reason for this lack of interest was the absence of apparent problems, or of what both the public and professional groups accepted as problems.

Clinical criteria created sharp boundaries between health and disease; someone was considered ill when his response to medical tests fell within certain parameters which physicians had defined as characteristic of a particular condition. A material only had an 'effect' when it caused certain types of changes or changes of a particular intensity. Changes below the thresholds or in other areas were not, for clinicians, effects at all. This policy defined away many chronic conditions, placing them out of sight, hence out of mind. The appeals for stricter regulation of DDT and warnings of possible dangers were, for many physicians and laymen alike, things they could not take seriously.

Another reason was the success of this type of standard in combatting disease, industrial dangers, and public health hazards. Based on experience to the early 1950s, these standards were adequate. In the later 1960s, though, both Congress and the public were far more willing to consider the possibility that DDT might have long-term effects on humans, effects which would not be uncovered through industrial testing or convict studies. The discovery of chemical carcinogens which induced tumours years after exposure raised a particularly frightening danger, and Congressional reaction to tests by the National Institutes of Health on DDT in 1969 indicates that lay people in the United States were wary of such hazards. In addition, studies of radiation and tobacco smoke increasingly cast doubt on the idea that there was a threshold of exposure below which DDT would have no effect. In these, and other, cases, any exposure increased the danger. Finally, public health problems, particularly the thalidomide disaster, caused a searching re-examination of the standards used to prove a chemical safe for human use. Critics had complained that the test groups used to show the safety of insecticide

residues were not representative of the general population, and that tests on experimental animals over their lifetime would be a better guide. By the late 1960s the American public was much more receptive to that argument than it had been in either 1935 or 1951.[9]

A New Problem: Insecticides and Wildlife

The reassessment of DDT in the late 1960s was only partly the result of new information and experiences which undermined accepted criteria of safety. A much more important factor was the discovery of previously unsuspected effects on wildlife, which not only stimulated new interest in studies on humans but provided the major part of the environmentalists' case for banning the chemical. Research on the environmental effects of DDT, though, was fundamentally different from that on man. Medical scientists had had available an alternative research strategy from the beginning of their work on DDT, and delay in recognizing the full effects in man can be attributed to their reliance on tests on exposed populations and concentration on acute, rather than chronic, effects. Wildlife biologists, on the other hand, had no directly relevant experience which would have suggested a research strategy; earlier pesticides had already been used on farmland, not in areas which would put the chemical in contact with wildlife. The delay in recognizing the full effects of DDT in the environment was due to another set of factors.

One was the speed with which DDT was introduced and spread. There was virtually no production of the chemical in 1943, but by the end of 1945 American industry was manufacturing over two million pounds a month. It was very inexpensive, and was so toxic to many insects (while relatively non-toxic to mammals) that it could economically be used over large areas where other insecticides were too expensive, and in places (such as suburbs) where earlier chemicals had been too toxic. Military use of DDT provided both a body of experience and surplus equipment for civilian use. Even before the end of World War II, economic entomologists had begun aerial spraying programmes against important forest insects — the gypsy moth and the spruce budworm — and to plan control campaigns in forests, swamps, and other previously unsprayed wild areas. In addition, towns and cities throughout America began to use DDT to kill mosquitoes, gnats, and the bark beetles which carried Dutch elm disease. Wildlife biologists faced an enormous and urgent

problem: to determine the effects of the new chemical under all these diverse conditions. And they had to concentrate, at first, on the immediate problems.[10]

The American agency with the major responsibility in this area was the Department of the Interior's Fish and Wildlife Service, which became involved in studying the side effects of forest sprays of the chemical in 1944. Its first problem was to determine the impact of forest sprays and to recommend practices which would kill the pests but not other forms of life. The agency's scientists concentrated on organisms which were known to be particularly sensitive to DDT or would be particularly exposed to it. They studied the effects on birds (particularly nestlings, who would be likely to consume large amounts of contaminated food) and on fish and marine invertebrates (which could tolerate only minute doses of the chemical). They found that, with suitable precautions, sprays would kill forest insect pests without producing unacceptable levels of mortality among non-target species. Applicators, they warned, must avoid streams, use minimum doses, leave areas unsprayed as refuges, and avoid spraying during the nesting season.[11]

The Fish and Wildlife Service continued to study the effects of insecticide sprays on wildlife, and to support research on these problems by scientists outside the agency. During the early 1950s, however, they were almost entirely concerned with local effects — immediate mortality in sprayed areas, and changes in wildlife populations exposed to repeated sprays. The Service had no mandate to engage in general oversight and regulation of insecticides in the environment, nor the funds to do so. It did not seek new problems but 'emphasized the experimental validation of field-developed hypotheses.'[12] In other words, it waited until problems developed and then investigated them.

The direction and extent of research clearly reflected Congressional and public priorities, as well as scientific estimates of where damage was most likely. In the immediate post-war period there was enormous public pressure in America to make DDT available and to use it, and a corresponding pressure on agencies checking on the chemical to finish studies as quickly as possible. The obvious problem was the immediate mortality from such programmes, and what resources the Service could devote to insecticides in the environment went, reasonably enough, to that question. There was, as well, no research to suggest that there might be more serious problems, and

no experience which would have led biologists to go looking for such difficulties. That a chemical might have properties that would make it a hazard to wildlife far from the point of application seemed, in the early 1950s, a hypothetical possibility so far-fetched as to be preposterous. Lacking clear evidence of such a hazard, scientists were reluctant to devote scarce resources to long-range research. Like medical scientists, wildlife biologists had a set of political, bureaucratic, and financial pressures that directed their attention to short-term problems, at the expense of long-range or fundamental work.

Early Warning Signals

The Fish and Wildlife Service's lack of authority to pursue a broad programme of environmental monitoring, and the general belief among scientists that there was no need for such investigations, meant that scientists only became concerned when environmental changes had become quite extensive. What aroused both scientific and public interest in the United States in the effects of DDT on non-target organisms, though, was not major environmental damage but high wildlife mortality in the wake of intensive sprays. The widespread use of DDT to control the bark beetles which carried Dutch elm disease caused the most obvious damage. The Department of Agriculture had first recommended DDT to control Dutch elm disease in the late 1940s and, despite modifications in the programme, it had continued to kill birds. The public, while it wished to keep its elm trees, often protested about the mortality among songbirds. The effects also troubled many scientists, and the programme gave Rachel Carson the image of the deathly village of *Silent Spring*.[13]

One of the scientists who became concerned about DDT because of the Dutch elm disease programme was Joseph J. Hickey, Professor of Wildlife Management at the University of Wisconsin, Madison. Since Hickey played an important role in the scientific work on the effects of DDT in the environment, and testified against the chemical at state and national hearings, it is worth considering the development of his ideas. In the late 1950s, as more and more communities in the Midwest turned to DDT to control Dutch elm disease, Hickey heard complaints about massive songbird kills following these sprays. He dismissed the reports, thinking that they

were exaggerated. He knew any insecticide would kill some songbirds if applied in heavy doses, but that the mortality would not, in the long run, have any effect on the population. Then, in the summer of 1958, a curious incident aroused his interest. Attending a funeral in a small Illinois town, he noticed a mulberry tree in the churchyard, loaded with ripe fruit. There was not a bird in sight. At the cemetery, outside town, he found the usual birds in their usual numbers. He questioned the townspeople. Yes, they told him, they had sprayed the town to control Dutch elm disease. Yes, there had been birds in town; they had come in the early spring, but had all gone. The people thought the birds had simply travelled further north, but Hickey wondered.

He decided to look into the matter. The following spring, with the aid of a grant from the Department of the Interior, he compared the songbird populations in three Wisconsin communities that had sprayed their trees with populations in three which had not. There was a disturbing pattern: a strong inverse correlation between the number and strength of insecticide sprays used and the songbird population. In some cases songbirds were almost completely absent. Shorewood, a suburb of Milwaukee, had a robin population 98 percent lower than in comparable towns which had not used DDT. 'Shorewood,' Hickey remarked some years later, 'had experienced *Silent Spring*.'[14]

That same year the campus of the University of Wisconsin, Madison, was treated with DDT, and Hickey observed the results. Analysis of dead birds, gathered with the help of the Buildings and Grounds Crew, showed that they had died of DDT poisoning. A comparison of the campus with a similar unsprayed area of the city showed a minimum kill of 86-88 percent among nesting species. The sprays had a continuing impact. Some species never returned to campus, and others re-established their populations only after a lapse of several years.[15]

Other scientists came to similar conclusions about the Dutch elm disease control programme on the basis of their own observations. George J. Wallace, an ornithologist at Michigan State University, began an investigation of the effects of the sprays on the campus at East Lansing in 1956. He and his students collected mortality figures from all over the state, worked out analytical techniques to measure concentrations of residues in organs, and undertook an extensive sampling programme. Charles Wurster, who helped form the Environmental Defense Fund and was its scientific coordinator, studied

the effects of Dutch elm sprays in New Hampshire. In 1958 Rachel Carson, alarmed and dismayed at the mortality among birds, began work on the book which appeared as *Silent Spring*.[16]

Two other extensive spraying programmes played important roles in focusing American public and scientific attention on the possible dangers of persistent pesticides in the environment. One was the use of DDT against the gypsy moth in the northeastern part of the United States, which federal and state authorities started in 1957. Both the public and scientists objected to the sprays, and one group on Long Island, led by a prominent zoologist, brought an unsuccessful suit to stop the work. The other instance was the use of poison bait containing dieldrin to combat the fire ant in the southeast. The damage to wildlife and commercial fisheries from these treatments was so great that several southern states withdrew from the cooperative arrangements under which the work was being done, and in 1958 public concern about the fire ant programme caused Congress to appropriate funds for environmental monitoring. This marked the beginning of large-scale coordinated efforts to assess the distribution of insecticide residues in the environment, and to measure their impact on various forms of life.[17]

The massive spraying programmes showed that the new insecticides could kill wildlife and underlined the need for caution, but few scientists argued for a ban on the chemicals. Even the most cautious thought the situation justified concern, not action. In 1960 studies of another kind raised new questions about the safety of insecticides to wildlife. Between 1948 and 1957, several large waves of mortality among adult birds wiped out a breeding colony of Western grebes at Clear Lake, California, about 100 miles north of San Francisco. Unable to find disease or poison, scientists investigating the grebes' death eventually analyzed the breast fat of some of the dead birds, and found very high levels of DDD (a chemical closely related to DDT). DDD had been applied to the lake to kill the larvae of the Clear Lake gnat in 1948, 1954, and 1957, shortly before each wave of mortality, but the chemical had been diluted to one part in seventy million, and had caused no observable ill effects on the lake biota. Scientists finally concluded that the pesticide had caused the grebes' death through bioconcentration, a buildup of residues through the food chains in the lake. A composite sample of plankton had 5.3 ppm DDD, 265 times the concentration in the lake water. Herbiverous fish had concentrations twice as high as that, and predaceous fish and birds had levels up to eighty five thousand times

that of lake water.[18]

These findings disturbed biologists far more than mortality from the Dutch elm disease programme had done. Deaths in the latter had occurred in restricted areas as the result of abnormally heavy sprays or through the massive, direct contamination of the birds' food supply. At Clear Lake, though, the insecticide had been applied in extremely low concentrations and had had no apparent ill effects. But instead of degrading or otherwise disappearing, the residues had persisted, and their solubility in lipid tissues had caused the bioconcentration which had killed the grebes. This phenomenon raised ominous possibilities. If bioconcentration was occurring on a large scale, any application of DDT, DDD, or similar compounds to the environment would add to a permanent burden on all organisms. Entomologists and wildlife biologists alike had assumed that discriminate uses of insecticides, uses which did not cause immediate mortality, were safe. The Clear Lake research suggested that there might be no possible discriminate use of these compounds.[19]

The Clear Lake studies were not the only evidence that the new insecticides might have hitherto unsuspected properties and effects in the environment. Work done at Patuxent Wildlife Research Center by the Fish and Wildlife Service had shown that repeated sprays depressed breeding populations of songbirds, and scientists had confirmed this phenomenon in the laboratory. In 1956, DeWitt found that the chlorinated hydrocarbon insecticides affected reproduction and reproductive success in captive quail populations.[20] Field observations suggested that this might be happening in the wild. By 1960, serious amateur ornithologists had noted population declines in several American species, notably the bald eagle population of Florida and the ospreys of Long Island Sound. Some observers linked the declines to sterility induced by environmental concentrations of insecticides. Fragmentary analysis showed suspicious correlations between high residue levels and chick deaths and non-viable eggs — but there was not enough data to settle the problem, merely to raise the question.[21]

A World-Wide Problem

Of more importance in shifting attention from studies of small areas to the general effects of insecticides in the ecosystem was the world-wide population crash of the peregrine falcon, an event one biologist

termed 'one of the most remarkable recent events in environmental biology.'[22] As early as 1960, rumours about widespread reproductive failure among populations of several birds of prey in eastern North America circulated at ornithological conferences, but there were no scientific studies showing the extent of the damage, if any. In the summer of 1963, Derek Ratcliffe, an English naturalist, published a report on the peregrine falcon in Great Britain which confirmed the rumours. The peregrine's population, usually stable, was falling and it was, he reported, not breeding in southern England. Even the isolated northern birds were experiencing reproductive failure. Ratcliffe found a suspicious correlation in both time and geography between these declines and the use of DDT in Great Britain and, drawing on DeWitt's work, he suggested that secondary insecticide poisoning was causing sterility and reproductive failures.[23]

Hickey had dismissed the earlier rumours, but after reading Ratcliffe's article, he decided to see if the same phenomenon was present in the eastern United States. In the spring of 1964 he sent two Wisconsin naturalists on a 14,000 mile journey from Georgia to Nova Scotia, checking 133 known peregrine eyries. All the sites had been active when Hickey had done a survey of eastern peregrines in 1939, and some were known to have been occupied since the 1860s. Hickey himself went to Europe to discuss the peregrine's situation with ornithologists there. Most of them confirmed Ratcliffe's description — broken eggs, dead nestlings, and a declining breeding population. His assistants had even more startling news for him on his return: they had found not a single viable fledgling in any of the nests. Their findings, Hickey said, were 'so startling as to make an international conference on the population biology of *Falco peregrinus* an absolute necessity.'[24]

The conference was held in Madison, Wisconsin, in the late summer of 1965 and was attended by more than 60 scientists. Several conclusions emerged from the debate. Comparing data they quickly established the dimensions of the problem: there was a world-wide population crash of peregrine populations in diverse ecological and geographical areas, a crash which followed a common pattern. It was clear that the usual causes — disease, local food shortages, human disturbance or killing — could not account for the phenomenon. Any single population decline might be explained; taken together they defied conventional explanation. There was, though, no consensus on the cause of the problem. Suspicion centred on the insecticides; DDT, in particular, was closely associated in

time and space with the population declines, had been found in the eggs and tissues of species which were failing to reproduce, and had been shown to be implicated in reproductive failure (though not in birds of prey). Some scientists were sure that the chemical was to blame. Others were less certain, pointing to ambiguities in the available data and to the paucity of information on insecticides in the environment, and on their effects on non-target organisms.[25]

The conferees did agree, though, that insecticide residues were the most likely cause, and they suggested several areas in which more research was needed. Far too little, they said, was known about the usual levels of insecticides and their metabolites in various species and the distribution of these compounds in various parts of the environment. No one knew what effects these compounds had in wildlife or, if they were causing reproductive failure, how they produced these effects. More tests were needed to see if the correlations between DDT and declining populations was real.

The peregrine's population crash was not the only environmental event to attract scientific attention, nor was the Madison conference the only meeting on the problem. Ratcliffe's article, the Clear Lake studies, and the work on bald eagle populations had generated concern in America and Europe. Two other conferences were held in 1965. On 30 May, biologists and ornithologists met in Port Clinton, Ohio, to discuss the population trends of the bald eagle; and in July, about 80 scientists met, under the sponsorship of the North Atlantic Treaty Organization, at Monks' Hole Experiment Station, in Great Britain. These conferences came to conclusions much like those reached in Madison. There were serious environmental disturbances; there was evidence that insecticide residues were implicated; but there was not enough field data or experimental work to satisfy most of the participants that the chemicals could be blamed for what was happening. There was a need for more routine collection of data on insecticides in the environment, for better analytical techniques in measuring residue levels, for more research on the dynamics of insecticides in various ecosystems, and for more coordination among various scientific specialties and between scientists in different countries.[26]

The recommendations for research pointed to the factors, most of them inherent in government and scientific organization, which had hindered the recognition of the problem: a lack of basic data on the fate of insecticide residues in the environment, and divisions across political and disciplinary boundaries. The orientation of earlier

research (which, as we have seen, had been away from basic, long-term studies) denied scientists the data they needed to assess the problem when they discovered it. Scientific specialization, though necessary, was a disadvantage in this case, where the problem did not fall clearly in one field. Scientists were working on different parts of the problem without being aware of all the relevant research. Only the references to analytical tools pointed to limitations in the available scientific knowledge as such.

Interdisciplinary Research

Between 1965 and 1968 scientists gathered new evidence, reassessed old data, and assembled information on the effects of DDT in the environment. It was an interdisciplinary effort, involving ornithologists, chemists, physiologists and many other types of scientists. Several separate lines of research converged — field studies of the affected species, studies of the distribution of pesticides in the environment and in food chains, physiological evidence on DDT's effects in mammals and birds, and laboratory studies growing out of field work. One new scientific development was important in this work: the vapour-phase chromatograph (VPC). Tracing the buildup of residues in the environment and analyzing for low concentrations with precision was, on any extensive scale, beyond the scope of the older methods of analysis. The standard Schecter-Haller test required a larger sample than the VPC, took more time and was specific for only one compound at a time. The VPC, which became commercially available about 1960, vastly simplified the task of analysis. It required only very small samples, was extremely sensitive, and identified all the components of the mixture at once. It was, perhaps, not essential to the progress of the work, but it was a major aid.

In both America and Great Britain scientists undertook studies of eggshell thickness in birds to determine if the suspected correlation of DDT and thin eggshells was real or an artifact of observation. Using private and museum collections, Ratcliffe in Britain and Hickey and Anderson in the United States traced the variation in weight of eggshells since the early 1900s, with strikingly similar results. On both continents there was a significant difference (about 19 percent) in the weight of peregrine egg shells before and after 1947 and a sharp drop in that year, just after the beginning of general use of

DDT. Nor was the pattern confined to a single species. Ratcliffe found similar changes in other birds of prey which had been reported as breaking their eggs, but no changes in ten other birds which did not have the problem. In the United States the eggs of Long Island ospreys, and of Florida bald eagles, showed the same changes.[27]

Scientists also accumulated data on the concentration of residues in various parts of the environment. Hickey, for example, undertook a study of bioaccumulation of DDT and DDE in Lake Michigan ecosystems, and measured the effects of the residues on herring gulls feeding in the area. (The Department of the Interior funded this work as part of its environmental monitoring programme.) He found the same pattern of bioaccumulation in food chains that scientists had found in Clear Lake, and a strong inverse correlation between the thickness of gull eggs and their level of DDE. 'The probability of this relationship occurring by chance,' Hickey said, 'proved to be 1 in 1,000.'[28] Other scientists did similar studies, giving, for the first time, an accurate picture of the extent of contamination in the environment.

Physiologists, working on the metabolism of drugs, added another link to the chain. In the early 1960s, a group investigating the metabolism of barbiturates in rats traced certain anomalous experimental results to the exposure of the test animals to chlordane, a chlorinated hydrocarbon insecticide similar to DDT. Further research showed that DDT, chlordane, and several other insecticides, accelerated the metabolism of barbiturates; they caused a proliferation of liver tissue which synthesized non-specific enzymes, which in turn broke down steroids.[29] In 1966, D.B. Peakall extended this work, showing that DDT and similar compounds produced the same phenomenon in birds. This was a key finding. The level of sex steroids in the female controlled the mobilization of calcium for the formation of eggshells. High levels of residues in the female lowered the level of the hormones in the bloodstream, causing thin shells.[30]

Wildlife biologists at the Patuxent Wildlife Research Center confirmed these results with another type of study. After the Madison conference, Lucille and William Stickel undertook feeding experiments on American sparrowhawks, birds closely related to the peregrine. Comparing the eggs of birds fed small amounts of DDT and dieldrin (another insecticide implicated in reproductive failure) with eggs from a control group on an insecticide-free diet, they showed that both chemicals caused thinning of shells and lowered reproductive success.[31] The Stickels' work linked field and laboratory

work, showing the cause and effect relation.

The combination of studies convinced many biologists that DDT and its metabolites, particularly DDE, were the cause of widespread reproductive failure in the peregrine and other birds of prey at the end of residue-laden food chains. In April 1968, as he prepared the proceedings of the 1965 conference for the printer, Hickey wrote that 'the ecological case against the chlorinated hydrocarbon insecticides . . . is essentially complete'.[32] Other scientists agreed. By then environmental groups were preparing for legal action against DDT, and scientists were closely involved with the effort. At hearings held between 1968 and 1972, far more scientists volunteered to testify against DDT than could be accommodated.[33]

DDT's effects on birds of prey was the most striking example of its action on non-target species far from any point of application, and the work on this problem was the clearest illustration of the evidence which brought many scientists to oppose the use of DDT. It was not, however, the only case. Scientists documented adverse effects of persistent chlorinated hydrocarbon insecticides on a variety of organisms in many different environments. They had found in fish, for example, reproductive failure, increased mortality of fry, behavioural changes, and increased susceptibility to environmental stress from low doses of the chemical. In ecosystems ranging from suburbs to salt marshes naturalists traced long-term damage to DDT residues.[34]

Conclusion

The scientific studies of DDT's effects in the environment provided the main impetus for a change in policy. Rachel Carson's *Silent Spring,* published in America in 1962, had converted the private scientific debate over the possible effects of DDT into a noisy public controversy, but did not significantly change either pesticide use or regulation. The bans on these chemicals in the United States were the result of the public acceptance of the scientific case against DDT, and of the use of scientific testimony to mount a legal challenge to the regulatory structure. In 1967 a new group, the Environmental Defense Fund, began legal action to halt the use of DDT. Drawing on the public for funds and on scientists for testimony, the group brought a series of lawsuits to halt use of DDT. By transferring the controversy from the agencies and their procedural mechanisms to

the courts, the EDF shifted the basis of the argument from compliance with the law to the adequacy of regulations and the effectiveness of government agencies in forming policy on the basis of scientific information.

The development of a DDT residue hazard (whether or not it was serious enough to warrant a ban on the chemical) was a failure of the regulatory process. Scientists did not quickly provide policymakers with the information necessary to make an informed decision about the costs and benefits of DDT use. The inadequacies of regulation have often been attributed to lack of funding for oversight, the lack of legal authority to stop hazardous practices, or to close relationships between the regulators and the regulated; and a full analysis of DDT policy, including the political and economic dimensions, would encompass all these factors. The case, though, illustrates another factor which logically precedes it: the inadequacy of scientists' experience as a guide to research strategy in a novel situation. In the case of human health it may be argued that the failure was not of science, but of a particular strategy; had the government relied on animal tests it would have uncovered much more rapidly the tumourogenic effects of DDT which led some oncologists to classify it as a potential human carcinogen. In terms of the environment, though, no such argument is possible. No one proposed studies which would have discovered DDT's bioconcentration, mobility, persistence, and hormonal effects, for no one thought such properties even remotely likely. Disciplinary boundaries, formed by experience to aid in the solution of problems, proved here to be a hindrance.

The normal problems of the regulatory process — funding, laws, and government-industry relationships — can, at least in theory, be solved. The inadequacy of experience as a guide, though, cannot. New technologies are, by definition, novel, and may present unusual hazards which will escape notice. We will, undoubtedly, continue to introduce new materials and inventions: and this means, necessarily, that we will continue to be exposed to the dangers of our ingenuity.

● NOTES

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